This project was intended to determine whether short-term exposure to moderate levels of photochemical pollutants affected the efficiency of various kinds of human motor performance. It does not appear that such performance is significantly altered by short exposures to moderate air pollution. Much more study is required.

AIR POLLUTION SIMULATION AND HUMAN PERFORMANCE

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SINCE the beginning of life, the atmosphere has been the repository for water and contaminants given off by human activities. Pollution of the air from man-made sources has increased sharply as population and urbanization have increased and our civilization has become more industrialized. The smog that hampers the human productivity of inhabitants in large cities such as Los Angeles now enlists the interest of both state and local governments. Cities such as Los Angeles suffer from the atmospheric presence of unburned hydrocarbons and nitrogen oxides which undergo photochemical changes after exposure to proper sunlight conditions with the resultant production of various nitrates, ozone, and other reactive pollutants. Most of the interest in the form of research has been directed toward air pollution control or studies of crop damage induced by smog.* Only a limited amount of research has been conducted in an attempt to determine the physiologic effects of air pollution on human beings.[23]

The immediate effects of air pollution are readily apparent in reduced visibility, as well as eye, nose, and throat irritations.[5] Balchum,[3] using volunteer emphysema patients for experiments, found that when they were breathing smoggy air, they were using more oxygen than when they were breathing filtered air. He also found that while the patients were consuming more oxygen, less of it was being transported through the arterial system. It is not yet clear from epidemiological studies whether there is a significant relationship between morbidity and mortality rates from chronic lung ailments and air pollution.[16,17,21,29] It has been rather well demonstrated, however, that exposure to even moderate levels of air pollution results in respiratory embarrassment and often dyspnea.[11,31] Coffin and Bloomer[6] recently reported that acute exposure of mice to high levels of automobile exhaust contaminants (100 ppm of carbon monoxide, oxidant range 35-0.67 ppm) caused enhanced mortality from streptococcal pneumonia.

Murphy, et al.,[19] exposed guinea pigs to variable levels of air pollution. In each case the animals demonstrated an increased expiratory flow resistance and decreased respiratory rate. Such findings suggest that physiological work effi-

* 4,9,12,13,15,16,22,27,30,32

ciency of animals or man would be significantly reduced as a result of exposure to air pollution contaminants.

Exercise and Air Pollution

It is generally recognized in areas such as Southern California that athletes competing on days of high smog irritation complain of pulmonary discomfort during and after the activity. Much of this discomfort may be psychological in nature. However, it would seem logical that there would be adverse physiological effects. The increased depth and rate of respiration in exercise, as well as changes in pulse rate and circulation, should cause more rapid dissemination of pollutants throughout the body, thus tending to increase any toxic effects.

Stokinger and his co-workers[28] demonstrated that physical activity may enhance the toxicity of ozone. Rats and mice were exercised in motor driven rotating cages and exposed to only 1 ppm of ozone. Fifty per cent of the exposed animals died within six hours. Unexercised rats, exposed to the same dosage for 200 days, showed no serious ill effects.

Smith[26] conducted the only published study on the effects of air pollution on human subjects who were exposed to the additional stress of exercise. In this experiment the subjects were exposed to an isolated constituent of photochemical air pollution—peroxyacetyl nitrate (PAN). The results of this study showed that 0.03 ppm of PAN significantly increased the metabolic oxygen cost of a moderate submaximum exercise task.

The purpose of the present study was to investigate the effects of simulated air pollution conditions on various motor and physiological parameters of human performance.

Design

In the summer of 1966, 14 college student volunteer subjects reported on two occasions to the Department of Engineering Air Pollution Test Facility at the University of California, Los Angeles. On each occasion the following parameters were measured: reaction time, vital capacity, and submaximum work performance on the bicycle ergometer. The subjects were randomly assigned to one of two groups according to the Latin square method of experimental design. With this design the subjects served alternately on the two occasions as either control subjects (performance in a normal atmospheric environment) or experimental subjects (performance in an air pollution environment). All data were analyzed for both experimental and treatment effects with the Analysis of Variance procedure for repeated measurements outlined by Winer.[33] The subjects were instructed that they were participating in a human performance study and apparently were not specifically aware of the concentration of pollutions in the air from the Air Pollution Chamber.

Air Pollution Simulation Facility

In order to simulate the conditions which take place in the outdoor environment of the Los Angeles basin, i.e., the ambient urban air, a test facility was designed and built as part of the Air Pollution Research Project of the University of California, Los Angeles. The details of the facility are described by Bush, et al.[4] A stationary automobile engine provides exhaust products which are delivered in appropriate amounts to a 6,000-cubic-foot tunnel or reaction chamber. The 200-foot-long tubular reaction chamber is constructed of four mill polyethylene walls which allow the exhaust constituents to undergo photosynthesis as a result of solar radiation (see Figure 1). In the present study the irradiated exhaust gases were pumped into an exercise booth near the reaction tunnel. A ventilation system was used to replace the exhaust gases

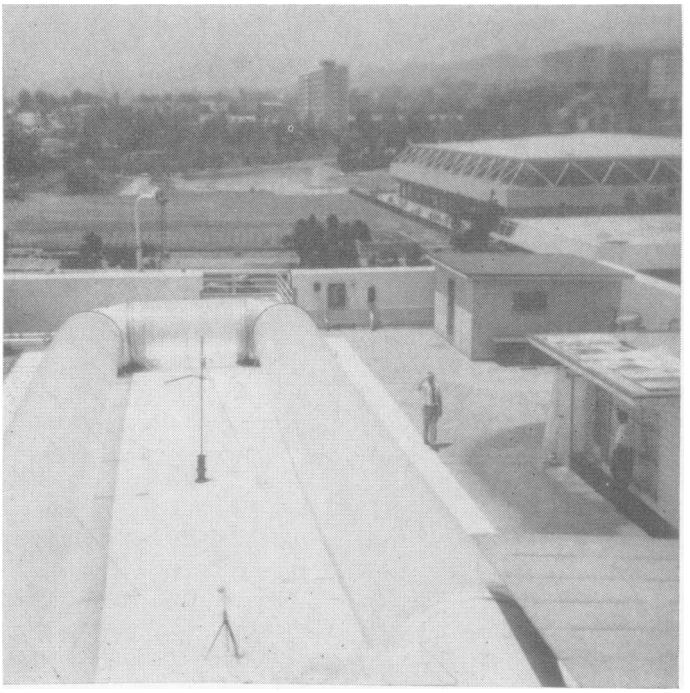

Figure 1—Air pollution reaction chamber

with filtered atmospheric air during the control experiments. Since the air introduced into the exercise booth was permeated with the odor of polyethylene during both control and experimental testing, it was assumed that this would tend to obscure the mild odor of the active constituents.

Air samples were drawn from the exercise facility and analyzed for the following contaminants: carbon monoxide, carbon dioxide, nitric oxide, nitrogen dioxide, oxidants, hydrocarbons, aldehydes, and formaldehyde. Carbon dioxide, carbon monoxide, and hydrocarbon were continuously monitored with nondispersive infrared analyzers. The nitric oxides and dioxides were continuously monitored by means of a Saltzman reagent in a Borman apparatus. Aldehyde and formaldehyde were collected in pitted glass absorbers and chemically analyzed with a 1 per cent sodium bisulfite reagent. Oxide analyses were conducted continuously, using alkaline KI solution in a Beckman apparatus. The exposure averages of these contaminants during the two days of experimental testing are shown in Table 1. The relative humidity of the exercise

Table 1—Exposure chamber contaminant concentrations

Agent	Quantity
CO	15–29 ppm
CO_2	800–1,400 ppm
NO	0.38–0.58 ppm
NO_2	0.7–1.00 ppm
HC_x	traces
Aldehydes	0.2–0.7 ppm
Formaldehyde	0.2–0.24 ppm
Oxidants	0.22–0.27 ppm

facility averaged 60 per cent, barometric pressure 29.6 inches of Hg, and air temperature ranged from 75° to 85° F. These environmental conditions simulate a moderate level of pollution in the Los Angeles basin.[4]

Performance Tests

The neuromuscular reaction time test utilized a Cramer 1/100 second clock with an audial stimulus and dominant index finger response. Standardized test instructions and procedures were followed as described by Atwell, et al.[2] Three test trials were administered and the best performance was used for purposes of statistical analysis.

The vital capacity test procedures followed in this study are outlined by Consolazio, et al.[7] A Collins six-liter water spirometer was used to administer three trials. In each trial the subject was instructed to exhale as much air as possible, following a maximum inhalation. A nose clip was used to prevent air leakage through the nasal passage. Thirty seconds of normal breathing were permitted between trials, and the best performance of the three trials was used for statistical analysis. Gaensher[8] and Worton, et al.,[34] have comprehensively described the clinical predictions which are based upon the vital capacity and maximum breathing capacity tests.

A modification of the submaximum work performance test developed by P. O. Astrand[1] was used for purposes of this study. In this procedure the subjects worked against an increasing work load on the bicycle ergometer until they achieved a heart rate of 150 beats per minute. Rate was maintained by means of an electric metronome at 50 cycles/min as resistance was increased 150 kilopond meters/min. (One kilopond= the force acting on the mass of one kilogram at normal acceleration of gravity. This measure of resistance multiplied by the distance pedalled=work rate in kilopond meters/min; 100 kpm/min=723 foot pounds/min.) Monitoring of heart rate was accomplished with a Sanborn ECG No. 100 cardiotachometer instrumentation using V 3 leads. Pulmonary ventilation was assessed throughout the exercise by means of a Kofranyi-Michaelis gasometer in circuit with a Hans Rudolph high velocity respiratory valve. An expired air sample was drawn from a plastic mixing chamber mounted above the gasometer at the completion of the work task, and analyzed for composition of O_2 and CO_2 by the Micro-Scholander[24] technic and the metabolic oxygen cost of the last minute of work computed. The bicycle work performance instrumentation is shown in Figure 2.

Results and Discussion

The general purpose of this project was to determine whether short-term exposure to moderate levels of photochemical pollutant constituencies affected the efficiency of various types of human motor performance. The analysis of the data is presented in Table 2. The daily, as well as the total experimental and control means, are shown for the five parameters of human performance. In addition, the data were arranged to allow an analysis of variance with the subsequent F test values being used to determine the statistical significance of the experimental and treatment differences.

It is readily apparent from comparison of the experimental means with the control means that exposure to a moderate level of pollution has little effect on the types of human motor performance chosen for this study. Average reaction time performance decreased .01 second. The work load required to achieve a heart rate of 150/b/min increased 89 kilopond meters. Although these above decrements in performance do suggest a deleterious effect from pol-

Figure 2—Bicycle work performance instrumentation

luted air, none of the F test values were statistically significant. The very small change in reaction time performance was not surprising since Xintaras[35] and others have amply demonstrated that at least a 5 per cent level of carboxyhemoglobin saturation is required before neurophysiological chemical inhibitions occur. These levels of blood contamination would not occur at the levels of air pollution imposed in the present study (CO=15-29 ppm). The subjects improved in vital capacity performance and metabolically required less oxygen to complete the bicycle work task following smog exposure, although these differences were not statistically significant either. Resting heart rate remained practically the same in the experimental and control environment.

The only significant difference observed in this study was due to a training effect. The work load required to achieve a heart rate of 150/b/min was significantly lower ($P<0.05$) on Day 1 than on Day 2 (MD=822 kpm). This apparent difference in physiological efficiency is probably just a reflection of differences in resting heart rate on the two days (MD=14.4 b/p/min). Although the differences in resting heart rate were not statistically significant (F value=1.13), it is logical to assume that the higher resting values on Day 1 would

Table 2—Analysis of variance-performance in air pollution and control environments

Test	Measure	Exper. mean		Total exper. mean	Control mean		Total control mean	F test		Significance
		Day 1	Day 2		Day 1	Day 2		Train. effect	Exper. effect	
Reaction time	Seconds	0.19	0.16	0.18	0.16	0.18	0.17	2.0	0.50	No No
Vital capacity	Liters	4.71	4.61	4.66	4.51	4.64	4.58	0	2.50	No No
Work performance	Kilopond meters	2,706	3,845	3,276	2,935	3,439	3,187	4.98	0	<0.05 No
Oxygen consumption	Liters/ min.	1.38	1.27	1.31	1.32	1.43	1.38	0	1.0	No No
Resting heart rate	Beats/ min.	84.5	88.2	86.5	95.0	78.5	86.7	1.13	0	No No

NOTE: This table should be read as follows. The experimental mean for reaction time (during exposure to an air pollution environment) was 0.18 and the control mean was 0.17. The comparison of reaction time performance on different days (training effect) resulted in an F of 2.0 which was not significant at the 0.05 level of confidence. The comparison of reaction time performance in a polluted environment as contrasted with normal air (experimental effect) resulted in an F of 0.50 which was not significant at the 0.05 level of confidence.

be reflected in a smaller work load required to achieve a working heart rate of 150/b/min. The higher resting heart rates on Day 1 probably reflected the subjects' apprehensiveness upon their first visit to the experimental laboratory. Such a psychological training effect could be avoided in future studies by providing a general laboratory orientation session for all subjects prior to the initiation of any data collection. Such a procedure was not possible in the present study due to the limited availability of the University Air Pollution Research Facility.

Probably the most important finding of this study, at least from a physiological point of view, was that work performance efficiency was not significantly altered by the air pollutants exposure. The mean work load required to achieve a heart rate of 150/b/min in a control environment was 3,187 kpm as contrasted with an experimental mean of 3,276 kpm. In addition the same subjects consumed only 1.31 liters O_2/min during the last minute of the bicycle work bout as compared with a mean control value of 1.38 liters O_2/min.

These findings do not corroborate the recently published results of a similar study by Smith.[26] Smith reported that exposure to the PAN (peroxyacetyl nitrates) constituencies of air pollution resulted in a significant increase in the oxygen uptake during a submaximum bicycle exercise task. The difference in findings might be due to the larger work load at which Smith's subjects pedalled. He reported oxygen consumption rates of 2.11 liters/min which are considerably higher than the rates of the present study. Since Smith did not report working heart rate values it was not possible to equate the cardiovascular component of physiological work efficiency. If, as Nadel and Comroe[20] theorize, inhalation of air pollutants results in bronchiolar constriction and increased airway resistance, then such an effect

would obviously be compounded during heavy exercise due to the decrease in the conductance-thoracic gas volume. Followed to its logical conclusion this theory suggests that respiratory and metabolic efficiency during exposure to contaminants would be most critically affected during maximum work bouts.

Future studies are needed to determine the effects of various levels of pollution exposure on MWC (maximum work capacity) and maximum oxygen uptake. In addition, further studies on human pulmonary function seem to be indicated. Undoubtedly the levels of pollution in the present study were not high enough to result in a significant respiratory irritant effect, since there was no significant change in vital capacity function. Future studies involving higher levels of contamination with more precise measures of airway resistance, such as expiratory flow rate and maximum breathing capacity, are recommended.

Summary and Conclusions

Within the limitations of this study it does not appear that the performance of fine neuromuscular tasks, e.g., reaction time, or cardiorespiratory work efficiency are significantly altered by short-term exposure to moderate levels of air pollution. Much more study, however, is required to elucidate the effects of air contaminants on other types of human psychomotor performance, especially maximum work capacity. It may well be that many atmospheric pollutants have an insidious qualitative biochemical effect on human physiological processes which can only be identified through careful longitudinal study. Such study should incorporate the use of repeated exposure technics to assess possible cumulative effects.

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Environmental Control in Hospitals

The Training Program of the National Communicable Disease Center will present a course in Environmental Control in Health Care Facilities, October 14-18, in Atlanta, Ga. The course is designed for public health workers and others responsible for curbing infection through environmental control activities in hospitals and similar institutions.

Further information can be obtained by writing to Chief, Community Services Training Section, Training Program, Room 414 Buckhead, National Communicable Disease Center, Atlanta, Ga. 30333